

BSG 2016 - Abstract Submission

Inflammatory Bowel Disease

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INFLUENCE OF IRON SUPPLEMENTATION ON THE NATURAL HISTORY OF COLITIS

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Introduction: Iron deficiency anaemia is common in IBD. Iron supplementation may induce or exacerbate colitis in rats (*APT* 2001;15:1989-99). Dysbiosis of the microbiota is common in IBD and iron contributes to this as it is a growth factor for pathogenic bacteria. We investigated the effect of dietary iron supplementation and/or reduction on the severity of chronic colitis in a murine model using clinical, histological and biochemical parameters.

Methods: Studies were performed on 6 groups of 8 wild type (WT) C57BL/6 mice. Chronic colitis was induced with 1.25% Dextran Sodium Sulphate (DSS) for 5 days, followed by 16 further days on water [for 3 consecutive cycles]. DSS-treated mice were fed one of three diets: low iron [LI] (100ppm), normal iron [NI] (200ppm) and high iron [HI] (400ppm) supplemented chow. Also, 3 non-DSS-treated groups were studied and fed similarly. Half of the mice in each control group were treated with 1 cycle of acute 2% DSS for 5 days at day 53, followed by 5 further days on water. All mice were sacrificed at day 63. Daily weights and clinical features were recorded. Histological colitis was scored using the Bauer score (*Gut* 2010; 59:1192-99). Faecal calprotectin was measured by ELISA and faecal iron by immunoassay at various time points [day (d) 1, 21, 42 & 63]. Statistical analyses used the Kruskal-Wallis test with post-hoc analysis.

Results: Oral DSS administration induced colitis in all treated mice. While chronic DSS colitis was not associated with weight loss, there was severe weight loss in acute DSS mice which was greatest in the low iron diet group ($p=0.001$ LI vs. HI; $p=0.01$ for LI vs. NI). Histologically, the colitis features were more prominent in acute DSS-treated mice ingesting low and high iron diets, with median colitis scores 6 & 5.5 respectively. Cyclic administration of DSS in drinking water resulted in a significant rise in faecal calprotectin, from baseline to d63 in LI ($p=0.05$) and for HI ($p=0.01$), but this was not significant in the NI group. In acute DSS, the rise was greater in LI and HI ($p=0.001$) and less in NI ($p=0.01$). Total faecal iron was increased in a dose-dependent manner within 9 weeks in all non-DSS groups. Nevertheless, in chronic DSS groups, $p=0.001$ at d1 vs. d63 for all groups [382% change for LI, 331% for NI and 355% for HI]. However, in acute DSS $p=0.05$ for LI, $p=0.001$ NI & HI (d1 vs. d10).

Conclusion: Changes in nutritional luminal iron exacerbate colitis. Oral administration of DSS causes a reproducible acute colitis, followed by a slow recovery phase with a concomitant chronic inflammation. Chronic colitis was worse in mice fed low or high iron diets, as shown by elevated calprotectin. Faecal iron rose equally in all 3 groups: with iron increases likely arising from diet and bleeding during colitis. Dysbiosis may be a consequence of this change in luminal iron.

Disclosure of Interest: None Declared